1: FEBS Lett 1994 Jan 10;337(2):200-6

Related Articles, Nucleotide, OMIM, Protein, NEW Books, LinkOut

Molecular cloning of a novel protein-tyrosine phosphatase containing a membrane-binding domain and GLGF repeats.

Maekawa K, Imagawa N, Nagamatsu M, Harada S.

Shionogi Institute for Medical Science, Osaka, Japan.

A full-length cDNA encoding a novel cytosolic protein-tyrosine phosphatase (PTP), PTP-BAS, was cloned from human basophils. Due to in-frame deletions in the coding region, PTP-BAS exists in three isoforms: 7,455 bp (2,485 aa) for type 1, 7,398 bp (2,466 aa) for type 2 and 6,882 bp (2,294 aa) for type 3. All three isoforms contain a single PTP catalytic domain at the carboxyl termini as well as two distinct structural sequences. Amino terminal sequences of 300 amino acids are homologous to membrane-binding domains of cytoskeleton-associated proteins. Three 90 amino acid internal repetitive sequences are homologous to the GLGF repeats found in guanylate kinase proteins. PTP-BAS was expressed in various human tissues, especially highly in the kidney and lung. Interestingly, the BAS mRNA level in the fetal brain was remarkably high.

PMID: 8287977 [PubMed - indexed for MEDLINE]

1: Cancer Res 1996 Jun 15;56(12):2742-4 Related Articles, NEW Books, LinkOut

IA-2, a transmembrane protein tyrosine phosphatase, is expressed in human lung cancer cell lines with neuroendocrine phenotype.

Xie H, Notkins AL, Lan MS.

Laboratory of Oral Medicine, National Institute of Dental Research, NIH, Bethesda, Maryland 20892-4322, USA.

IA-2 is a transmembrane protein tyrosine phosphatase isolated recently from a human insulinoma subtraction library. Its expression in normal human tissues is restricted primarily to the pancreatic islets and brain. In this report, we describe the expression of IA-2 mRNA in a panel consisting of 20 lung tumor cell lines with neuroendocrine and non-neuroendocrine phenotype and 17 non-lung tumor cell lines. IA-2 mRNA was detected in 8 of 11 neuroendocrine small cell lung carcinomas, 4 of 4 non-small cell lung carcinomas with neuroendocrine phenotype, and 11 of 12 non-lung neuroendocrine tumor cell lines. In contrast, IA-2 mRNA was not detected in five non-neuroendocrine lung carcinomas, nor in a panel of other non-neuroendocrine tumor cell lines. The expression pattern of IA-2 mRNA suggests that IA-2 may represent a new marker for neuroendocrine differentiation In human lung cancer cells and perhaps other neuroendocrine tumors.

PMID: 8665506 [PubMed - indexed for MEDLINE]

1: Cancer Lett 1995 Jul 13;93(2):239-48 Related Articles, NEW Books, LinkOut

SCIENCE DIRECT FULL-TEXT ARTICLE

Increased mRNA expression of the receptor-like protein tyrosine phosphatase alpha in late stage colon carcinomas.

Tabiti K, Smith DR, Goh HS, Pallen CJ.

Cell Regulation Laboratory, National University of Singapore.

The protein tyrosine phosphatase alpha (PTP alpha) mRNA level in paired samples of late stage (Dukes' D) colorectal tumors and adjacent normal colon mucosa was quantified by RNase protection assays. After normalization against 18S RNA or beta-actin mRNA level, a 2-10-fold increase in PTP alpha mRNA was detected in 10 of 14 tumors (approximately 70%) compared to mucosa. In situ hybridization of digoxigenin-labelled antisense PTP alpha RNA to tumor and mucosa sections produced a signal only in neoplastic cells of the tumor sample, consistent with the high increase in PTP alpha mRNA detected by RNase protection assays of some of the tumors. This is the first report suggesting an association of a protein tyrosine phosphatase with colorectal carcinoma. PTP alpha is a receptor-like PTP thought to be involved in regulating cell proliferation. Its oncogenic properties when overexpressed in cultured fibroblasts suggest that PTP alpha overexpression could contribute to the tumorigenic process in colon carcinoma.

PMID: 7621435 [PubMed - indexed for MEDLINE]

1: Anticancer Res 1996 Mar-Apr;16(2):943-6

Histochemically demonstrable protein tyrosine phosphatase in human breast and colorectal cancer: large decrease in its activity in colorectal cancer suggests a tumor suppressor role in colorectal mucosal cells.

Partanen S.

Department of Pathology, Jorvi Hospital, Espoo, Finland.

Many oncogene products and growth factor receptors are protein tyrosine kinases, and exert their cellular effects by the phosphorylation of tyrosyl residues of effector proteins. The balance and dynamic renewal of phosphotyrosine proteins are also regulated by protein tyrosine phosphatases (PTPs), whose inhibition under experimental conditions causes cellular proliferation and transformation, with a concomitant increase in phosphotyrosine protein content. Inverse effects are obtained by increasing PTP activity. On the basis of these effects, PTPs might also function as tumor suppressors in human tissues. This possibility was further investigated here by demonstrating PTP and phosphotyrosine protein content with histochemical techniques. In normal human breast tissue PTP activity was low and in the majority of breast cancers the activity was increased and exhibited great variation between different cases. When the relationship of phosphotyrosine protein content with PTP was evaluated, no inverse dependence was detected, suggesting that in human breast tissue and cancer PTP may not show tumor suppressor activity. In normal colorectal mucosae PTP activity was high, while in all colorectal cancers it was very low, constituting only 14% of the activity present in normal mucosal cells. The great drop in PTP activity together with reported alterations in a gene encoding a PTP and in a chromosome containing a PTP gene in colorectal cancer strongly suggest that PTP may function as a tumor suppressor in human colorectal mucosae. The decrease in PTP activity may be one factor stimulating or causing neoplastic proliferation in multistep colorectal carcinogenesis.

PMID: 8687156 [PubMed - indexed for MEDLINE]

1: Cell Signal 1996 Nov;8(7):467-73 Related Articles, NEW Books, LinkOut

Suppression of insulin receptor activation by overexpression of the protein-tyrosine phosphatase LAR in hepatoma cells.

Li PM, Zhang WR, Goldstein BJ.

Dorrance H. Hamilton Research Laboratories, Department of Medicine, Jefferson Medical College of Thomas Jefferson University, Philadelphia, PA 19107, USA.

Protein-tyrosine phosphatases (PTPases) play an essential role in the regulation of reversible tyrosine phosphorylation of cellular proteins that mediate insulin action. In order to explore the potential role of the transmembrane PTPase (LAR) in insulin receptor signal transduction, we overexpressed the full-length LAR protein in McA-RH7777 rat hepatoma cells and found that modest increases in the abundance of LAR protein expression downregulated a number of insulin-stimulated cellular responses closely related to the activation of the receptor kinase. An increase in LAR protein of 2.4-fold over the level in control cells caused a 40% reduction in insulin receptor autophosphorylation in intact cells, without an alteration in insulin receptor mass or a change in the insulin-stimulated receptor kinase activity measured with partially purified receptors in vitro. In addition, insulin-stimulated tyrosine phosphorylation of the endogenous insulin receptor substrates IRS-1 and Shc were decreased to 57% and 73% of control, respectively, and IRS-1 associated phosphatidylinositol 3'-kinase activity was reduced to 47% of control of the cells overexpressing LAR. The present results, taken with our recent data demonstrating that reducing the abundance of LAR by expression of antisense mRNA enhances insulin receptor signal transduction (Kulas D. T., et al. J. Biol. Chem. 270:2435, 1995), supports the hypothesis that LAR acts as a physiological modulator of insulin action in insulin-sensitive hepatoma cells.

Publication Types:

- Review
- Review, Tutorial

1: J Biol Chem 1996 Nov 29;271(48):30916-21

Expression of the protein tyrosine phosphatase beta2 gene in mouse erythroleukemia cells induces terminal erythroid differentiation.

Kume T, Watanabe T, Sanokawo R, Chida D, Nakamura T, Oishi M.

Institute of Molecular and Cellular Biosciences, University of Tokyo, Bunkyo-ku 113, Tokyo, Japan.

We have cloned cDNA for protein tyrosine phosphatase beta2, which had been implicated in erythroid differentiation of mouse erythroleukemia cells. Expression of cDNA constructs, in which beta2 cDNA is placed under the control of mouse metallothionein-I promoter, by ZnCl2 converted a significant portion (20 to 38%) of the cells to erythroid-like cells, which is 25-50% of the erythroid differentiation efficiency observed by conventional erythroid-inducing agents. Furthermore, introduction and expression of altered protein tyrosine phosphatase beta2 cDNA constructs designed to produce the enzyme lacking the phosphatase activity inhibited erythroid differentiation by 100-20%, depending upon the concentration of erythroid-inducing agents employed. These results strongly suggest that protein tyrosine phosphatase beta2 is involved in triggering erythroid differentiation in mouse erythroleukemia cells.

PMID: 8940077 [PubMed - indexed for MEDLINE]

Entrez PubMed

1: J Biol Chem 1999 Mar 19;274(12):7958-68

Related Articles, NEW Books, LinkOut

PubMed Services

Calcium/calmodulin-dependent phosphorylation and activation of human Cdc25-C at the G2/M phase transition in HeLa cells.

Patel R, Holt M, Philipova R, Moss S, Schulman H, Hidaka H, Whitaker M.

Department of Biochemistry, University of Leicester, University Road, Leicester, United Kingdom LE1 7RH.

Related Resources

The human tyrosine phosphatase (p54(cdc25-c)) is activated by phosphorylation at mitosis entry. The phosphorylated p54(cdc25-c) in turn activates the p34-cyclin B protein kinase and triggers mitosis. Although the active p34-cyclin B protein kinase can itself phosphorylate and activate p54(cdc25-c), we have investigated the possibility that other kinases may initially trigger the phosphorylation and activation of p54(cdc25-c). We have examined the effects of the calcium/calmodulin-dependent protein kinase (CaM kinase II) on p54(cdc25-c). Our in vitro experiments show that CaM kinase II can phosphorylate p54(cdc25-c) and increase its phosphatase activity by 2.5-3-fold. Treatment of a synchronous population of HeLa cells with KN-93 (a water-soluble inhibitor of CaM kinase II) or the microinjection of AC3-I (a specific peptide inhibitor of CaM kinase II) results in a cell cycle block in G2 phase. In the KN-93-arrested cells, p54(cdc25-c) is not phosphorylated, p34(cdc2) remains tyrosine phosphorylated, and there is no increase in histone H1 kinase activity. Our data suggest that a calcium-calmodulin-dependent step may be involved in the initial activation of p54(cdc25-c).

PMID: 10075693 [PubMed - indexed for MEDLINE]

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     (FILE 'HOME' ENTERED AT 19:40:07 ON 04 APR 2002)

     FILE 'MEDLINE, BIOSIS, CANCERLIT, LIFESCI, BIOTECHDS' ENTERED AT
19:40:55
     ON 04 APR 2002
L1            11 S PTP(W)(10 OR X)
L2             0 S TYROSINE(W)PHOSPHATASE(W)(10 OR X)
L3         14890 S TYROSINE(W)PHOSPHATASE
L4           280 S L3(5A)(RAT#)
L5             6 S L1 AND PY<1998
L6             4 DUP REM L5 (2 DUPLICATES REMOVED)
L7           179 S L3(3A)(RAT#)
L8           133 S L7 AND PY<1998
L9           122 S L8 AND L3/TI
L10           59 DUP REM L9 (63 DUPLICATES REMOVED)
L11         1802 S L3(S)RAT#
L12            4 S L11 AND (PLOWMAN?/AU OR JALLAL?/AU)

=> log h
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WEST Search History

DATE: Thursday, April 04, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=JPAB,EPAB,DWPI; PLUR=NO; OP=ADJ			
L7	L6 and (testis or testicular)	0	L7
L6	L5 and rat	13	L6
L5	tyrosine adj phosphatase	249	L5
DB=USPT; PLUR=NO; OP=ADJ			
L4	L3 and testi$5	9	L4
L3	L1 with rat$1	32	L3
L2	L1 with rat	28	L2
L1	tyrosine adj phosphatase	460	L1

END OF SEARCH HISTORY